eration of Goll's columns. In descending direction there was degeneration in Lowenthal's descending marginal fascicle of the anterior column and in the intermediary fascicle of the lateral column (faiscean intermediaire du cordon lateral of Lowenthal).

2. On the sixth day beginning degeneration of the lateral cerebellar tracts (Kleinhirnseitenstrangbahn) was present in addition to the degenerations mentioned under 1.

3. On the 14th day degeneration of the lateral pyramidal tracts is noticed. The highest intensity of florid decay of the medullary sheaths is reached on the 4th day in Lowenthal's fascicles, on the 12th to 14th days in Goll's columns and the cerebellar tracts.

4. The descending degeneration of the posterior columns at the locality of Schultze's comma is to be seen on the 4th day in slight extent; it does not increase later. The temporal succession of the degeneration in the various tracts accordingly corresponds to that of the development of medullary sheaths. Those tracts of the fibres of which receive their medullary sheaths first degenerate first. From Flechsig's researches it is known that the fibres of Goll's columns are supplied with medullary sheaths at the end of the sixth month, those of the cerebellar tracts (Kleinhirnseitenstrangbahn) towards the beginning of the seventh and the pyramidal tracts towards the end of the ninth month, or in other words the same temporal succession as they degenerate. ONUF.

Acute Mania.—DeWitt (*Lancet-Clinic*, June 22, 1895). The medical treatment of these cases is very simple, and can be disposed of in few words. To procure sleep and quiet is perhaps the greatest desideratum, and I know of nothing so certain in its action as chloral hydrate, given in 40 or 60 grains It may be given alone or combined with one of the bromides. Bromidia I have always found very reliable. It is almost certain to quiet and produce sleep. Occassionally one meets with cases that resist the influence of chloral even in large repeated doses; here opium or some of its derivatives either given alone or in connection with the chloral, will be found of service. If hypodermically administered, not less than $\frac{1}{8}$ gr. should be given. Small doses only excite the patient, and do more harm than good. Hydrobromate of hyoscine has some advocates. The milder hypnotics, such as sulfonal, chloralamid, etc., are not to be thought of in these cases; they are practically inert, and do no good. J. C.

PATHOLOGICAL.

The Pathological Anatomy of Chronic Chorea.—*Gazzetta degli Ospedali e Delle Cliniche*, Mar. 16, 1895. At a meeting of the R. Academy of Medicine of Rome, February 25, 1895, Dr. Bignami reported the results of his pathological studies on two cases of chronic chorea in adults, without hereditary history. In one case he found sclerosis of the cerebral arteries, slight thickening of the pia, and atrophy of the gyri in the frontal and parietal lobes, which presented microscopically multiple foci of disseminated chronic encephalitis.

In the other case he found no alterations of the meninges, atrophy of the convolutions, numerous miliary patches, sub-cortical of sclerosis especially in the frontal lobes, a large focus of sclerosis in the external nucleus of the thalamus which diffused itself into the sub-thalmic region interrupting somewhat the radiating fibres of the internal capsule; much smaller foci were also found bilaterally and symmetrically in the red nucleus. Microscopically these foci are composed of a fine fibrillary tissue not in nuclei; cellular elements are also present having large rounded or oval nuclei, and abundance of protoplasm. KRAUSS.

Vertigo.—One of the most valuable contributions to our knowledge of the puzzling symptom known as vertigo has recently come from the pen of Mendel of Berlin. He says that various theories have been formulated by medical men on the subject of vertigo, and it would be well, to come to an understanding as to what this condition really is. In the first place, vertigo is almost invariably ushered in by ocular phenomena. In some cases the patient has the sensation of seeing the surrounding objects whirling around himself, and experiences a tendency to fall either in the same direction as the objects seemingly turn, or toward the other side; at other times, it seems to the patient as if the objects about him were approaching or receding, rising or falling.

This instability of the external world is immediately followed by a sensation of oppression and sinking, resulting in static disturbances, and the patient begins to stagger and collapse. Then follow various secondary phenomena, such as pain in the back of the head, noises in the ears, ephemeral deafness, vomiting, slowness of the pulse, diaphoresis, etc.

There are four degrees of vertigo; in the first and lightest form, only ocular symptoms occur, accompanied by angor which rapidly disappears; in the second, there is a sensation of disturbed equilibrium; in the third, the equilibrium is really upset and the patient falls; in the fourth, these symptoms are accompanied by the above-mentioned secondary phenomena.

The pathognomonic clinical sign of vertigo is disturbance of the equilibrium. Maintenance of the equilibrium is intimately connected with the sense of touch, kinæsthetic sensations, and sight. Whether hearing has anything to do with it, is still a subject of controversy.

All static disturbances are not symptomatic of vertigo Abolition of the sensorium results in a loss of the equilibrium, which has nothing in common with vertigo; the fear experienced on being placed at a considerable height is not vertigo properly so called, but rather a psychical process which only secondarily affects the organs of equilibrium; the same is true of agoraphobia.

True, vertigo is alwavs associated with a disturbance in the musculature of the eyes. Vertigo is the first symptom complained of by patients who suffer from paralysis of the ocular muscles, whether this paralysis be peripheral or nuclear. On several occasions I have even seen vertigo precede the paralysis.

On investigation of the causes of ocular vertigo, with special reference to the supply of blood to the muscles of the eye, I have found that the nuclei which preside over the movements of the ocular muscles receive their blood from the posterior cerebral artery through fine terminal branches, not anastomosing with each other. The result of this arrangement is that the slightest disturbance in the central circulation immediately detracts from the supply of blood to the nuclei of the ocular muscles.

Every pathological process which develops a focus of disease in the brain will necessarily influence the cerebral circulation and, in consequence thereof, be associated with vertigo, especially when this focus occupies a posterior cerebral fossa.

Lastly, vertigo is the earliest and most prominent sign of a morbid change in the blood vessels of the brain, although arteriosclerosis is not accompanied by vertigo unless its evolution is very rapid, a process of adaptation, taking place otherwise.

The treatment of vertigo must be adapted to its cause. In cerebral arteriosclerosis iodide of potassium in small doses, continued for months, and, in addition, camphor and quinine, to re-enforce the action of the heart, seems to give the best results. With the same object in view, alcoholic beverages in moderate quantities should be prescribed to patients who suffer from this affection. In recapitulation he says, vertigo consists in a group of symptoms, determined by a disturbance in the sense of equilibrium, due to defective function of the ocular muscles. This defect itself may be of peripheral or central origin. J. C.